

Measuring of the extent of atherosclerotic lesions in the human by means of the assessment of the degree of activation of blood coagulation factor VII

Citation for published version (APA):

Muller, A. D., Gonggrijp, R., & Hemker, H. C. (1975). Measuring of the extent of atherosclerotic lesions in the human by means of the assessment of the degree of activation of blood coagulation factor VII. *Artery*, 1(4), 366.

Document status and date:

Published: 01/01/1975

Document Version:

Publisher's PDF, also known as Version of record

Please check the document version of this publication:

- A submitted manuscript is the version of the article upon submission and before peer-review. There can be important differences between the submitted version and the official published version of record. People interested in the research are advised to contact the author for the final version of the publication, or visit the DOI to the publisher's website.
- The final author version and the galley proof are versions of the publication after peer review.
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Download date: 05 May. 2023

MEASURING OF THE EXTENT OF ATHEROSCLEROTIC LESIONS IN THE HUMAN
BY MEANS OF THE ASSESSMENT OF THE DEGREE OF ACTIVATION OF BLOOD
COAGULATION FACTOR VII

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Factor VII is the coagulation factor that is activated by the contents of body cells. The activated factor VII (factor VIIa) in its turn activates factor X. Factor Xa together with factor V forms the prothrombin-converting enzyme, thus being responsible for the formation of thrombin that plays a key role in thrombus formation.

It appears that factor VII activation is not a simple pro-enzyme - enzyme transition. Intermediate activation stages do occur. Native factor VII (factor VII_n) is converted into a state with enhanced activity (factor VII_e) that is transformed in fully activated factor VII (factor VII_a) which is quickly inactivated (factor VII_i).

The products of the reaction sequence
factor VII_n - factor VII_e - factor VII_a - factor VII_i
each have their own half-life in the circulation. We developed methods to assess the forms n, e, and a separately.

We tested the hypothesis that cell wall lesions cause a partial activation of factor VII. To this end we assessed the form and degree of activation of factor VII in groups of patients known to have extensive atheromatous degradation of the intima of major arteries. One control group existed of normals, another of patients with generalized carcinomatous disorders.

It appeared that the difference between cell wall lesions by atheromatous degeneration and carcinomatous infiltration is reflected in the state of activation of factor VII.